Advances in Computer Sciences

The Stochastic Model

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Introduction

Recent research has revealed a surge in the application of Stochastic Differential Equations (SDEs) in the modeling of infectious diseases. Factors emanating from this surge has been the ability of stochastic differential equations to lay one of the most imperative fundamental differences in the asymptotic dynamics of the deterministic and stochastic epidemic models [1]. These fundamental differences include the convergence of the sample path of stochastic models to the disease-free state and that of the deterministic model to the endemic equilibrium. Stochastic phenomenon occurs naturally in our environment, and when small number of reacting molecules is involved in the modeling system, deterministic models become inappropriate [2]. Stochastic models have properties which include the probability of an outbreak, the quasistationary probability distribution, the final size distribution of an epidemic and the expected duration of an epidemic that makes it unique from others [3]. Stochastic Processes can have moments and covariance function associated with them, which are functions of time, as do random variables [4].

Stochastic epidemic models have been studied extensively, and the outcomes have been astonishing as they have provided an alternative means of investigating epidemic outbreaks in a small populations coupled with better elucidating of the transmission dynamics of epidemic outbreaks in the early stages of an epidemic outbreak, insinuating the robustness of the deterministic model with respect to stochastic perturbations [5]. Kiouach and Omari [6], considered an epidemic model of type SIRS and the stability of the interior equilibrium point was discussed. The model was modified to stochastic version and the stability properties of the stochastic model were investigated. Maroufy et al [7], studied a classical model of a SIRS epidemic in an open population. They deduced an explicit formula by which the lower bound of the density of the infective can be computed. The Model was then extended to stochastic version and Lyapunov functional was employed to investigate the global stability of both the deterministic and the stochastic model. Lahrouz et al [8], presented an epidemic SIRS model with saturated incidence rate and disease-inflicted mortality. The model was modified into stochastic version and the global stability of the models were studied using the tool of Lyapunov function. Further, the global existence and positivity of the solution was verified as well as the global stability in probability proved under suitable condition by the perturbation of white niose. Murkherjee [9], considered deterministic and stochastic models for prey-predator system where the prey population is infected by micro parasite. The stochastic stability properties of the models were investigated which suggested the robustness of the deterministic model to stochastic fluctuations. Clancy [10], formulated a stochastic SIS epidemic model for transmission of infectious disease through a population, considering direct host-host transmission and indirect transmission through free-living infectious stages. Guoting and Tiecheng [11], investigated a stochastic version of the SIR model. The stability in probability of the steady state of the system was proved under favorable conditions with white noise perturbations. Mukherjee et al [12], studied the behaviour of a plant herbivore for both discrete and continuous model with stochastic perturbation. The existence and stability of two fixed points were investigated which suggested local asymptotic stability in probability for the stochastic model for certain strengths of white noise. Bifurcation diagrams and time series plots were obtained for the model.

Entrenched in these works are devotees of outstanding achievements and contributions towards scientific community on the mathematical modeling of infectious diseases. In this section, the dynamics of deterministic model of malaria disease and its stochastic variant are presented. The stochastic version of the model is derived by introducing random perturbation to the equations of system (1). Numerical examples are provided to illustrate the explicit differences between the deterministic model and the stochastic system.

The Stochastic Epidemic Model Formulation

The stochastic model of the deterministic system (1) is obtained by introducing stochastic perturbations in the main parameters of the deterministic system. Thus we permit stochastic perturbations of the variables $S_H, E_H, I_H, R_H, S_V, E_V, I_V$ around

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their values at positive equilibrium E* [13,14,15]. Further, we assume that the white noise of the stochastic perturbations of the variable around values of E* are proportional to the distances $S_H, E_H, I_H, R_H, S_V, E_V, I_V$ from $S_H^*, E_H^*, I_H^*, R_H^*, S_V^*, E_V^*, I_V^*$. Hence, the stochastic version of model (1) is given by

$$dS_H = \left[\pi_H + \rho_H R_H - \sigma_V \beta_H \frac{I_V}{N_H} S_H - \mu_H S_H \right] dt + Q_1(S_H - S^*_H)dB_1$$

$$dE_H = \left[\sigma_V \beta_H \frac{I_V}{N_H} S_H - (\nu_H + \mu_H)E_H \right] dt + Q_2(E_H - E^*_H)dB_2$$

$$dI_H = \left[\nu_H E_H - (\gamma_H + \mu_H + \delta_H)I_H \right] dt + Q_3(I_H - I^*_H)dB_3$$

$$dR_H = \left[\gamma_H I_H - (\rho_H + \mu_H)R_H \right] dt + Q_4(R_H - R^*_H)dB_4 \quad (1)$$

$$dS_V = \left[\pi_V - \sigma_V \left(\frac{\beta_V I_H + B_{VH}R_H}{N_H} \right) S_V - \mu_V S_V \right] dt + Q_5(S_V - S^*_V)dB_5$$

$$dE_V = \left[\sigma_V \left(\frac{\beta_V I_H + B_{VH}R_H}{N_H} \right) S_V - (\nu_V + \mu_V)E_V \right] dt + Q_6(E_V - E^*_V)dB_6$$

$$dI_V = \left[\nu_V E_V - \mu_V I_V \right] dt + Q_7(I_V - I^*_V)dB_7$$

With Q_i, for = 1, 2 ... ... 7 are real constants and B_i= 1, 2, 3, 4, 5, 6, 7 are independent wiener processes.

Numerical Method for SDEs-Euler-Maruyama

This section focuses on stochastic modeling application and numerical simulations of trajectories. The work employs the fundamental works which allow the development of effective numerical methods for stochastic differential equations (SDEs), as well as providing the basic information necessary to understand the nature of SDEs and how to stimulate them by using the tool of Euler-Maruyama method [16,17,18]. The method has been proved to have a strong global convergence under the assumption that the stochastic differential equation is locally Lipschitz and that the moment of the exact and numerical solution are bounded for some p > 2.

Consider a scaler, autonomous SDE in integral form

$$X(t) = X_0 + \int_0^t f(s,x(s))ds + \int_0^t g(s,x(s))dw(s), \ 0 \le t \le T \quad \textbf{(2)}$$

Where f and g are scaler functions and the initial condition x_0 is a random variable.

If x(t) is the solution to (2), then the solution x(t) is a random variable that arises when we take the zero step size limit in the numerical sense. Hence, the differential equation form of (2) can be written as

$$dx(t) = f(t, x(t))dt + g(t, x(t))dw(t), \ \text{with X(0)=0} \le t \le T \quad \textbf{(3)}$$

Now, from equation (3), if g= 0 and x_0 is constant, then the problem becomes deterministic and (3) reduces to autonomous ordinary differential equation

$$\frac{dx(t)}{dt} = f(t,x(t)), with \ \text{X(0)=} x_0 \quad \textbf{(4)}$$

Applying Euler-Maruyama method to (3) over (0, T), we first discretize the interval by letting $\Delta t = \frac{T}{L}$

for some positive integer L and T_j= $j\Delta t$. Hence, our numerical approximation to X (T_j) will be denoted by X_j. Therefore, the Euler-Maruyama (EM) method becomes

$$X_j = X_{j-1} + f(X_{j-1})\Delta t + g(X_{j-1})(W(T_j) - W(T_{j-1})), for \ \text{j=1,2,....L} \quad \textbf{(5)}$$

Equation (5) arises from the integral form

$$X(T_j) = X(T_{j-1}) + \int_{T_{j-1}}^{T_j} f(s,x(s))ds + \int_{T_{j-1}}^{T_j} g(s,x(s))dw(s) \quad \textbf{(6)}$$

Further, equation (6) reduces to Euler's method when g = 0 and x_0 is constant (see [19,20,21]).

Numerical Examples and Discussion

Here, we illustrate with figures the dynamics of the deterministic and stochastic systems, and gives an explicit difference in the models

by carrying out numerical simulation for hypothetical set of parameter values. To demonstrate the differences, we simulate the stochastic and the deterministic systems by using the set of parameter values in table 1. To confirm the deterministic plots, we choose arbitrary white noises $Q_1, Q_2, Q_3, Q_4, Q_5, Q_6$ and Q_7 of equal strength 0.5, and shows the fluctuations in the trajectories of the stochastic system. We can see that the trajectories of the stochastic plots displayed are the same as the trajectories of the deterministic model during a finite time frame. This suggest that even though fluctuation occurs, they result in the same behavior as the deterministic one. The deterministic approach has limitations that the stochastic handles in a more realistic way. Thus, the deterministic model gives the same results any time the simulation is run with the same parameter and initial values. This might be mathematically correct, however, this is not the case in real epidemic situation, as there may be many parameters which we cannot not model entirely realistically. Modelling them deterministically, we lose some of the complexity of the system. Hence, it is appropriate to assume a stochastic perturbation. Hence, the differences in the dynamics of the models are given by the figures 1-12.

Conclusion

In this section, the dynamics of the deterministic malaria model with standard incidence rate and its stochastic version are presented. The stochastic version of the model was constructed by employing stochastic fluctuations in the main parameter of the deterministic model. Our main purpose of the study was to investigate the dynamics of the deterministic and stochastic systems, by giving numerical examples to illustrate the explicit difference in the models. The numerical simulations for the models show that the trajectories of the stochastic plots were the same as the trajectories of the deterministic model. Further, from the stochastic plots, the simulations show

Table 1: Description of variables and parameters of the Malaria Model (1)

Parameter	Description	Estimated value	Reference
π_H	Recruitment rate of human	2.5	[9]
β_H	Transmission probability from an infectious mosquito to a susceptible human	0.9	[9]
β_v	Transmission probability from an infectious human to a susceptible mosquito	0.8	[9]
β_{vH}	Transmission probability from a recovered human to a susceptible mosquito	0.009	[9]
μ_H	Natural death rate of human	0.00004	[9]
δ_H	Death rate from the disease	0.00354	[9]
γ_H	Infectious human recovery rate	0.003704	[9]
V_H	Progression rate from EH to IV class	0.08333	[9]
V_v	Progression rate from EV to IV class	0.1	[9]
ρ_H	Rate of loss immunity in human	0.0146	[9]
σ_v	Recruitment rate of mosquitoes	500	[9]
σ_v	Biting rate of vector	2.9	[9]
μ_v	Natural death rate of vector	0.071 4	[9]
α_H, α_v	The level at which the force of infection saturates	(0,1)	[9]
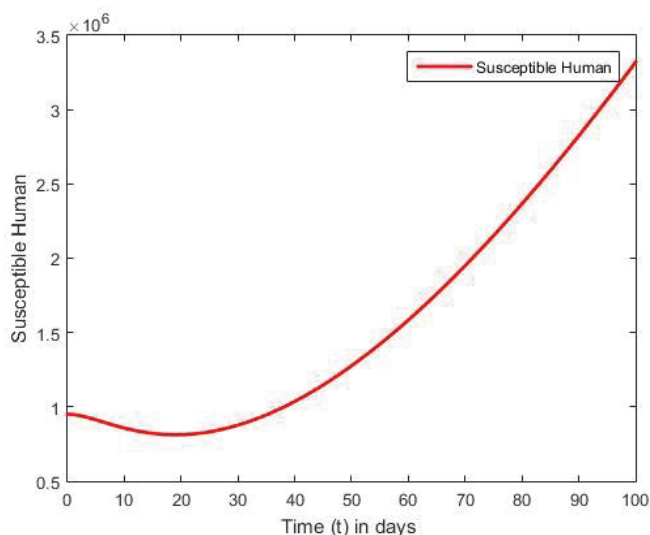
Figure 1: The plot represents population of Susceptible Human
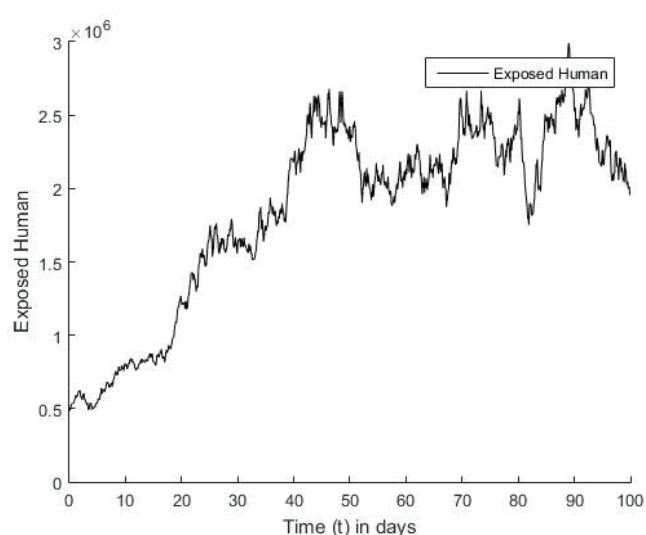
Figure 4: Stochastic plot of Exposed Human
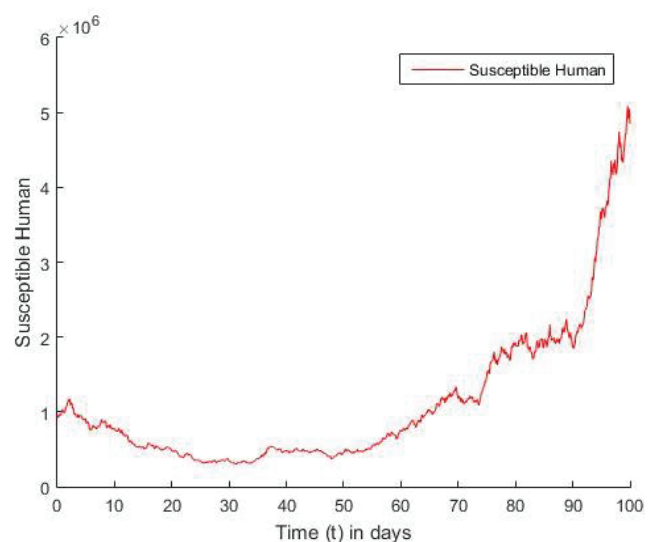
Figure 2: Stochastic plot of susceptible Human
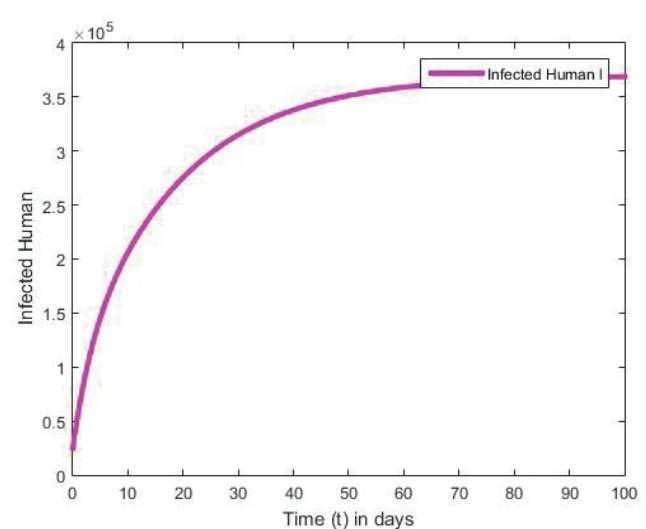
Figure 5: The plot represents population of Infected Human
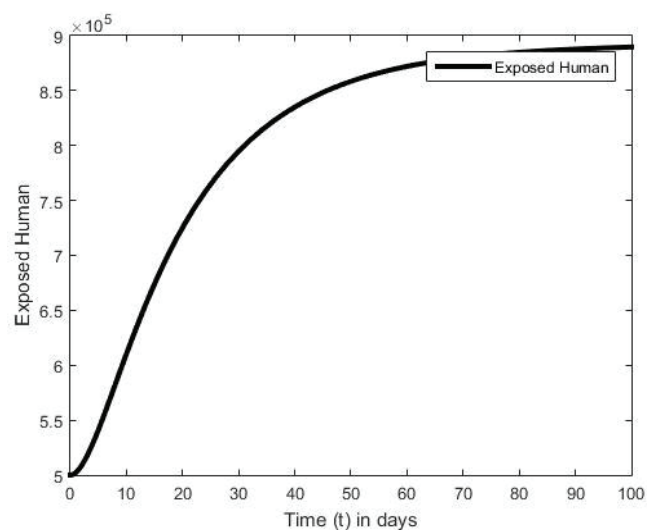
Figure 3: The plot represents population of Exposed Human
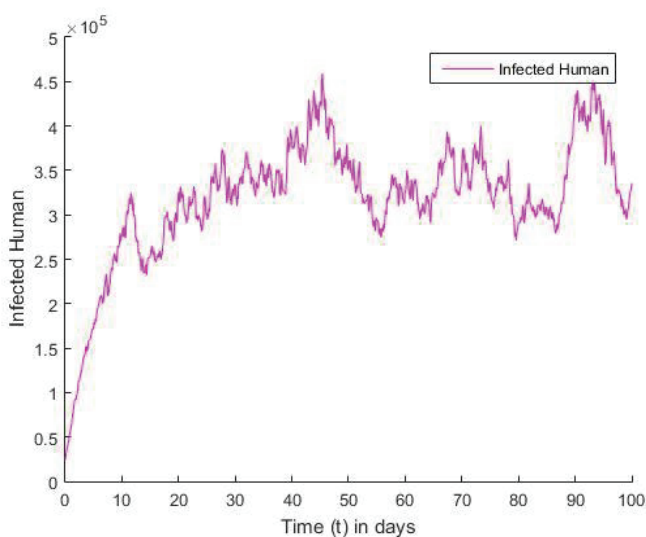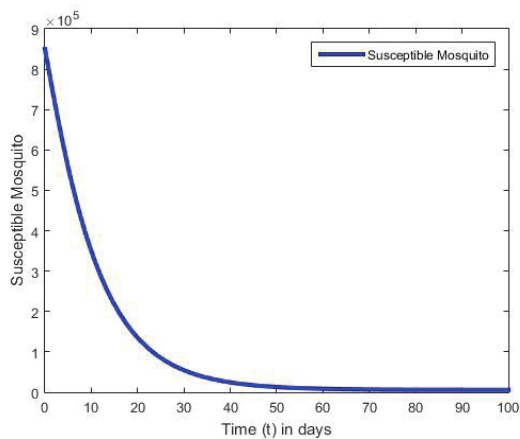

Figure 6: Stochastic plot of Infected Human

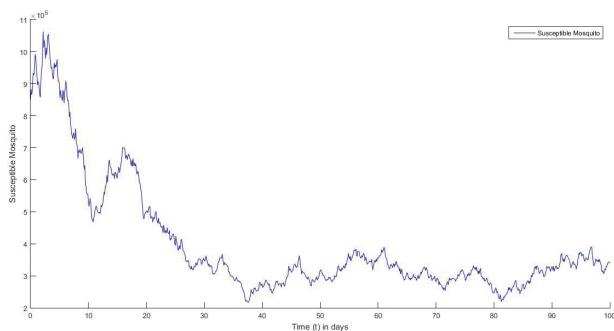**Figure 7**: The plot represents population of Susceptible Mosquito

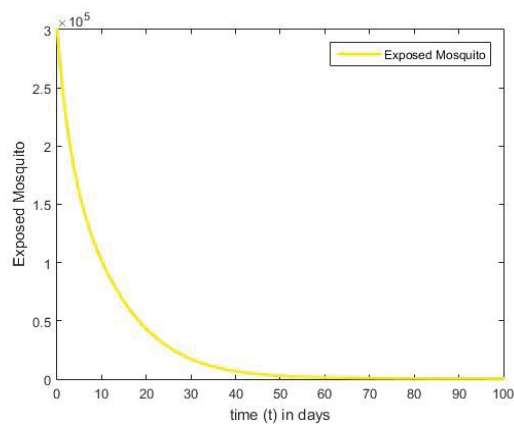**Figure 8:** Stochastic plot of Susceptible Mosquito

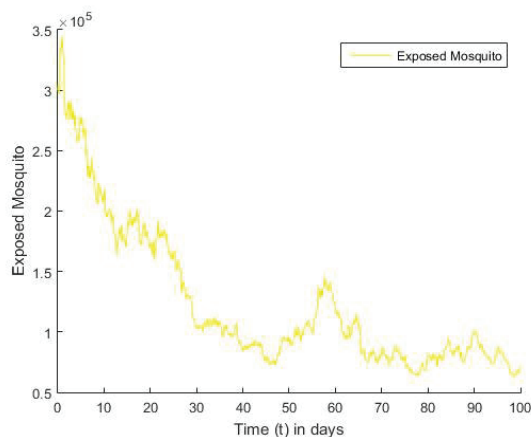**Figure 9:** The plot represents population of Exposed Mosquito

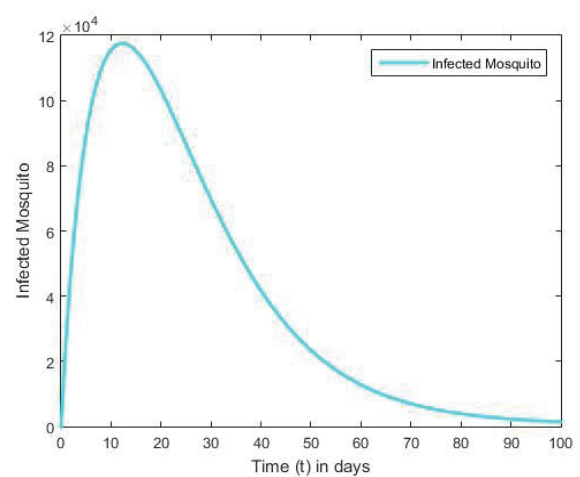**Figure 10**: Stochastic plot of Exposed Mosquito

Figure 11: The plot represents population of Infected Mosquito
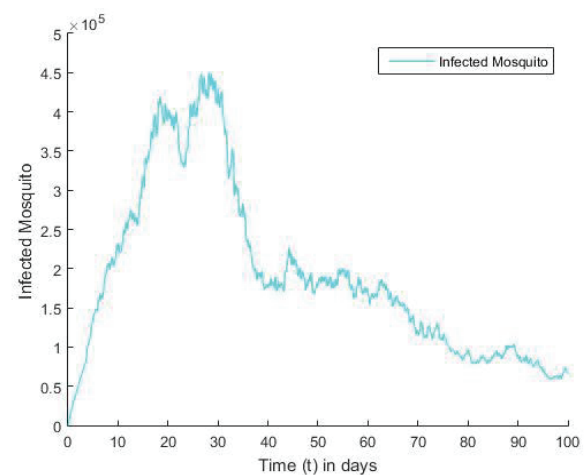

Figure 12: Stochastic plot of Infected Mosquito

an initial random fluctuation of the stochastic trajectories, until they eventually approach asymptotic level. The numerical example shows that the stochastic system is globally asymptotically stable in probability when the densities of white noise are less than certain threshold parameters. However, if these densities of white noise are zero, it means there are no stochastic environmental factors on the population and hence no stochastic perturbation.

References

1. Kunita H (1990) Stochastic flows and stochastic differential equations. Cambridge Studies in Advanced mathematics Vol. 24. Cambridge University Press.

2. Brauer F, Van den Driessche P, Wu J (2008) Mathematical Epidemiology. Mathematical Biosciences subseries. Springer-Verlag Berlin Heidelberg

3. Linda JSA (2008) An Introduction to Stochastic Epidemic models. Department of Mathematics and Statistics. Texas Tech University, Lubbock, Texas, 79409-1042

4. Burrage K, Burrage PM, Tian T (2004) Stochastic Analysis with Applications to mathematical Finance, Mathematical, Physical and Engineering Sciences 460: 373-402

5. Isham V (1991) Assessing the variability of Stochastic epidemics. Math Biosci 107: 209-224

6. Kiouach D, Omari L (2008) Stochastic Asymptotic Stability of a SIRS Epidemic Model. European Journal of Scientific Research 24: 575-579

7. Maroufy H El, Lahrouz A, Leach P (2011) Qualitative Behaviour of a Model of an SIRS Epidemic, International Journal of Applied Mathematics and Information Sciences 5: 220-238

8. Lahrouz A, Omari L, Kiouach D (2011) Global analysis of a deterministic and stochastic nonlinear SIRS epidemic model. Nonlinear Analysis: Modelling and Control 16: 59-76.

9. Mukherjee D (2003) Stability analysis of a stochastic model for prey-predator system with disease in the Prey. Nonlinear Analysis: Modelling and Control 8: 83-92

10. Clancy D (2005) A Stochastic SIS Infection Model Incorporating Indirect Transmission. Journal of Applied Probability 42: 726-737.

11. Guoting C, Tieching L (2009) Stability of Stochastic Delayed SIR model. Stochastic and Dynamics 9: 231-252. doi: doi. org/10.1142/S0219493709002658

12. Mukherjee D, Das P, Kesh D (2011) Dynamics of a Plant-Herbivore Model with Holling Type II Functional Response. Computational and Mathematical Biology, Concept Press Ltd 2: 1-11

13. Das P, Mukherjee D, Hsieh YH (2011) An S-I epidemic model with saturation incidence: discrete and stochastic version. International Journal of Nonlinear Analysis and Applications 1-9

14. Liu XQ, Zhong SM, Tian BD, Zheng FX (2013) Asymptotic properties of a stochastic predator-prey model with Crowley-Martin functional response. Journal of Applied mathematics and computing 43: 479-490

15. Adnani J, Hattaf K, Yousfi N (2013) Stability Analysis of a Stochastic SIR Epidemic Model with Specific Nonlinear Incidence Rate. International Journal of Stochastic Analysis 2013: 1-4

16. Higham DJ, Mao X, Stuart A (2002) Strong convergence of Euler-type methods for non-linear stochastic differential equations. SIAM J Numer Analysis 40: 1041-1063

17. Platen E (1999) An introduction to numerical methods for stochastic differential equations. Acta Numerica 8: 197-246. doi: doi.org/10.1017/S0962492900002920

18. Kloeden PE, Platen E (1992) Numerical solution of stochastic differential equations. Springer

19. Higham DJ (2001) An Algorithmic Introduction to Numerical Simulation of Stochastic Differential Equations. Society for Industrial and applied Mathematics 43: 525–546

20. Maruyama G (1955) Continuous Markov processes and stochastic equations. Rend Circ Mat Palermo 4: 48-90

21. Burrage K, Burrage PM (2001) Order conditions of stochastic Runge-Kutta methods by B-series. SIAM J Numer Analysis 38: 1626-1646